Successful delayed bilateral renal revascularization during active phase of Takayasu's arteritis

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Successful bilateral renal revascularization was performed 24 days after the development of angiotensin converting enzyme-inhibitor–induced bilateral renal artery thrombosis and anuric acute renal failure in a patient with Takayasu's arteritis. Excellent results were obtained after an unusually long ischemic time for a patient with active-phase disease. The outcome suggests that aggressive surgical revascularization can benefit patients with renal failure caused by renal arterial occlusion during the active phase of Takayasu's arteritis. (J Vasc Surg 1998;27:552-4.)

Takayasu's arteritis is a rare large vessel inflammatory disease of unknown etiology, usually affecting women of childbearing age. Inflammatory lesions are usually present in arterial media, leading to progressive narrowing of major aortic branches at their orifices. Subclasses of Takayasu's arteritis can be grouped according to the location of lesions: class I affects the aortic arch and great vessels; class II affects the thoracoabdominal aorta; class III involves both the aortic arch and the thoracoabdominal aorta; and the unusual class IV (or IIb) affects isolated nonaortic large arteries, such as pulmonary, cardiac, or abdominal arteries.[1,2] Clinical features usually include both systemic symptoms, such as fever or musculoskeletal arthritis and pain, and symptoms due to vascular inflammation or ischemia referable to the anatomic location of lesions, most commonly carotid bruits, claudication, or diminished pulses.[2,3]

Renin-dependent hypertension is common, present in 30-60% of patients, and may be due to bilateral or unilateral renal artery stenosis.[3,4] Surges in plasma renin activity often lead to episodes of malignant hypertension, with consequent cardiac dysfunction and flash pulmonary edema.[5] Although the presence of renal pathology in Takayasu's disease confers significant morbidity that is not always surgically correctable, recent experience suggests that excellent results can be achieved with rapid diagnosis and prompt repair.[1-3,6,7]

We report a case of acute renal failure in a patient with severe active Takayasu's disease that was successfully revascularized after 24 days of renal artery occlusion. Because this unusually long period of ischemia was reversed we propose that patients with active Takayasu's disease may rapidly develop enough collateral circulation to sustain renal viability and that aggressive revascularization is indicated after control of uremia.

CASE REPORT

A 19-year-old white woman was transferred to the Johns Hopkins Hospital for management of malignant hypertension and acute renal failure of 3 days duration. The patient had a 4-year history of ankylosing spondylitis and an elevated erythrocyte sedimentation rate. One month before admission, she had fever, malaise, and severe hypertension. Blood urea nitrogen and serum creatinine levels were normal. Arteriography demonstrated severe proximal stenosis of the renal, celiac, and superior mesenteric arteries, and a thickened abdominal aorta (Fig. 1). The thoracic aorta was normal. The radiologic appearance was consistent with Takayasu's arteritis (class IV) but not with fibromuscular dysplasia.

The patient was treated with high-dose steroids for 1 week before transfer to our hospital. Three days before transfer she received two doses of the angiotensin converting enzyme (ACE) inhibitor enalapril because of worsening hypertension associated with generalized seizures. Immediately after drug administration there was a sudden drop in blood pressure to 130/70 mm Hg concomitant

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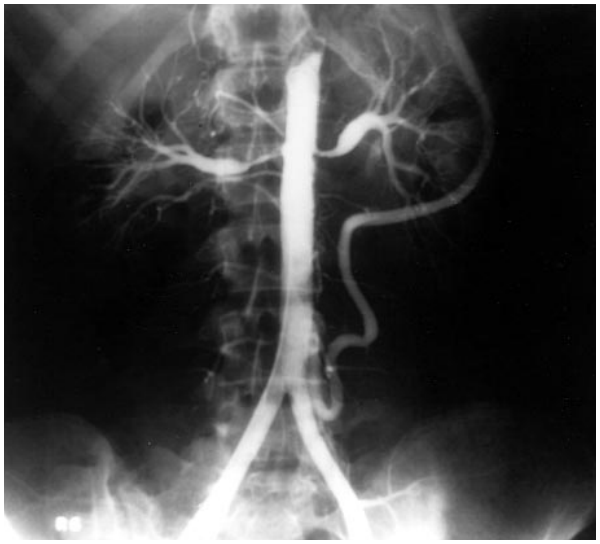

Fig. 1. Preoperative angiogram demonstrates bilateral proximal renal artery stenosis.

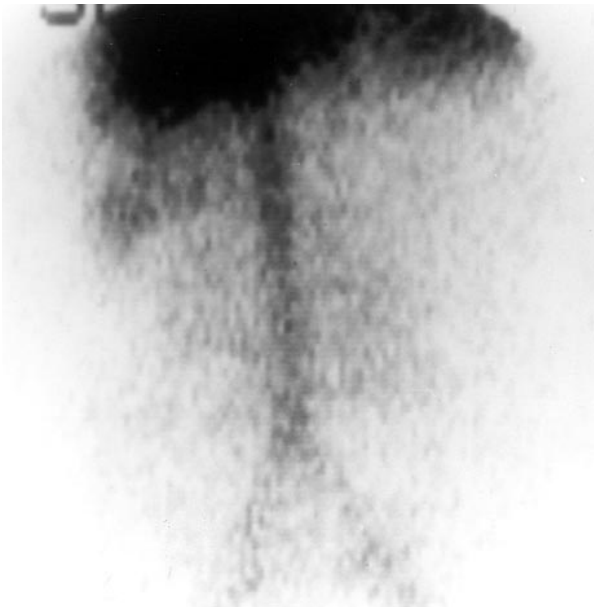

Fig. 2. Preoperative renal scan demonstrates bilateral loss of renal function.

with the onset of severe left flank pain. A renal scan 1 day before transfer demonstrated no flow or function in the left kidney (Fig. 2). The glomerular filtration rate of the right kidney was estimated to be 6 ml / min.

At transfer to our hospital, the patient's blood pressure was 210 / 110 mm Hg with normal fundi. There was a loud left abdominal bruit and marked left flank tenderness. There were normal peripheral pulses and no edema. A neurologic examination had normal findings. Blood urea nitrogen level was 84 mg / dl, and serum creatinine level was 6.4 mg / dl and rose progressively. Hemodialysis therapy was initiated 3 days after transfer. Because acute renal failure in this setting is usually reversible,[8] and renal revascularization is usually considered a high-risk procedure during the active phase of Takayasu's nephritis, immediate revascularization was not attempted. Progressively increasing doses of clonidine, nifedipine, and labetalol were required for blood pressure control. Blood pressure decreased to approximately 140 / 80 mm Hg, and surgical revascularization was scheduled. Prednisone therapy (1 mg / kg) was continued to treat the vasculitis.

Fourteen days after the onset of ACE inhibitor–induced acute renal failure, the patient's condition suddenly deteriorated after she received two units of packed red blood cells. The planned surgical revascularization was canceled. The blood pressure became labile with episodes of severe hypertension, seizures, global left ventricular dysfunction, and acute pulmonary edema, necessitating intubation. Intravenous antihypertensive therapy with nitroprusside, hydralazine, and clonidine was initiated. Continuous venovenous ultrafiltration and intermittent dialysis were required for control of pulmonary edema. Because of poor cardiac function (ejection fraction 18%), cardiac catheterization was performed. This demonstrated normal coronary anatomy without vasculitis. An ACE inhibitor was administered to control the life-threatening malignant hypertension. A second renal scan demonstrated some blood flow to the left kidney, but glomerular filtration rate remained less than 10 ml / min.

With evidence that neither kidney was completely infarcted, bilateral aortorenal arterial grafts with saphenous vein were placed 21 days after admission. Neither biopsy nor revascularization of lesions in the visceral vessels were performed because of the patient's precarious condition. Occluding left and partially occluding right renal arterial thrombus was removed. Renal arterial tissue showed no characteristics of vasculitis. The patient had an uneventful postoperative recovery; a renal scan 5 days postoperatively showed much improved flow to both kidneys. Antihypertensive therapy was rapidly tapered. Venovenous ultrafiltration was discontinued, and chronic intermittent hemodialysis was reinstituted. At discharge on postoperative day 12, blood pressure was 150 / 80 mm Hg with two antihypertensive medications.

Outpatient dialysis was discontinued after 3 months because of recovery of renal function, and antihypertensive medications were discontinued slowly. After 1 year, the patient has resumed all activities. She had normal renal function (blood urea nitrogen 10 mg / dl, creatinine 1.0 mg / dl; Fig. 3) and had normal blood pressure without antihypertensive medications.

DISCUSSION

This case demonstrates that successful renal revascularization is possible several weeks after development of anuric acute renal failure caused by renal arterial occlusion. Successful delayed renal

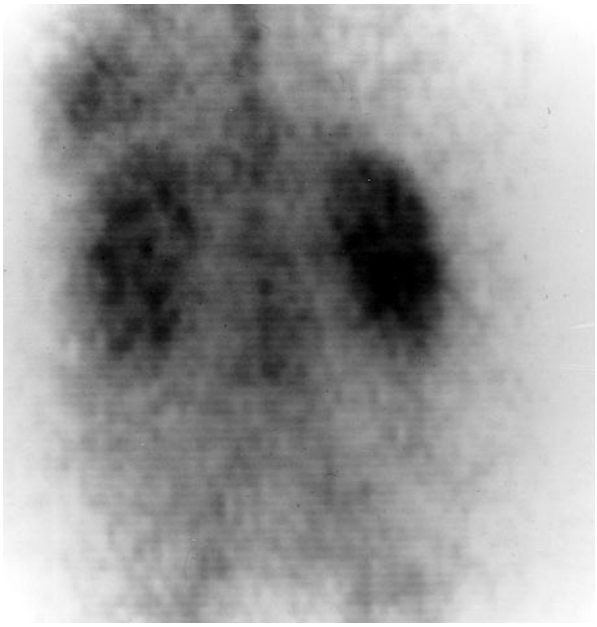

Fig. 3. Postoperative renal scan demonstrates restored bilateral renal function.

revascularization in patients with renal atherosclerosis or fibromuscular dysplasia has been reported.[9,10] One factor contributing to these good outcomes is the presence of collateral circulation around preexisting long-standing renal artery stenoses. Although this patient had an acute injury and no long-standing renal artery stenoses caused by fibromuscular dysplasia or atherosclerosis, collateral circulation was likely present, because hypertension, and thus hemodynamically significant renal artery stenoses, were present for at least 2 months before revascularization, and the patient was young. Collateral circulation most likely prevented complete renal infarction after the sudden, severe drop in blood pressure upon initial administration of the ACE inhibitor. However, we believe this insult was sufficient to produce bilateral renal arterial thrombosis. Otherwise, the ACE inhibitor–induced acute renal failure would have responded to discontinuation of the ACE inhibitor.[8]

This case is unique because surgical intervention was mandated during the acute phase of arteritis. There has been the suggestion that surgical procedures performed during the active phase of Takayasu's arteritis—defined by new or worsened systemic symptoms, elevated erythrocyte sedimentation rate, vascular ischemia or inflammation, or typical angiographic findings—have less optimal results than procedures performed when the disease is inactive (5-year patency 53% vs 88%).[3,6] For this patient malignant hypertension resulted in severe cardiac dysfunction with life-threatening flash pulmonary edema.[5] The initial decision was made to manage the life-threatening hypertension with ACE inhibitors, abandoning the kidneys. This therapy was partially successful, because the patient exhibited improved cardiac function for 1 week. An operation was then performed to salvage renal function and relieve heart failure. The likely absence of renal parenchymal disease evidenced by continued renal blood flow on a renal scan also favored revascularization. The choice to use saphenous vein as the bypass conduit was based on the patient's young age. Although it is possible for recurrent arteritis to develop in venous grafts, such progression of disease is unusual.[2,7]

Although renal angioplasty appears safe for patients with Takayasu's arteritis, the low success rate of angioplasty suggests that it may be useful only as temporary treatment.[2,3] In this disease marked by severe morbidity and disability from renovascular hypertension, aggressive surgical renal revascularization, even when delayed, may allow excellent surgical and functional results.

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